

Complete Atrioventricular Block Complicating Acute Myocardial Infarction in the Thrombolytic Era

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- OBJECTIVES** We assessed the incidence, associated clinical parameters and prognostic significance of complete atrioventricular block (CAVB) complicating acute myocardial infarction (AMI) in the thrombolytic era and compared them to data from the prethrombolytic era.
- BACKGROUND** The introduction of new therapeutic modalities to treat AMI, aimed to enhance coronary reperfusion and to limit myocardial necrosis, was expected to decrease the incidence of CAVB and to improve prognosis. However, there are only limited data regarding the incidence and the prognosis of AMI patients with CAVB in the thrombolytic era.
- METHODS** Data from 3,300 patients from the Israeli Thrombolytic Surveys (prospective, nationwide surveys of consecutive patients with AMI in all 25 coronary-care units in Israel in 1992 and 1996) were analyzed and compared with data from 5,788 patients included in the SPRINT (Secondary Prevention Reinfarction Israeli Nifedipine Trial) Registry (1981 to 1983).
- RESULTS** During the 1990s, the incidence of CAVB was 3.7% compared with 5.3% in the 1980s, $p = 0.0007$. In the 1990s, mortality of patients with CAVB was significantly higher than in those without CAVB at 7 days (odds ratio [OR] = 4.05 95% CI [confidence interval] 2.34 to 6.82, 30 days OR = 3.98 [95% CI 2.44 to 6.43] and one-year hazard ratio [HR] = 2.36, [95% CI 1.68 to 3.30]) and similar in thrombolysis-treated and not-treated patients. Mortality of patients with CAVB has not changed significantly between the two periods; seven-day OR = 0.82 (95% CI 0.46 to 1.43); 30-day OR = 0.78 (95% CI 0.45 to 1.33) and one-year HR = 0.79 (95% CI 0.54 to 1.56), respectively, in the 1990s as compared to a decade earlier.
- CONCLUSIONS** The incidence of CAVB complicating AMI is lower in the thrombolytic era than in the prethrombolytic era. Mortality among patients with CAVB is still high and has not declined within the last decade. The AMI patients who develop CAVB in the thrombolytic era have significantly worse prognosis than do patients without CAVB. (J Am Coll Cardiol 1999;34: 1721-8) © 1999 by the American College of Cardiology

Patients with an acute myocardial infarction (AMI) developing complete atrioventricular block (CAVB) in the prethrombolytic era fared worse during hospitalization than did patients without this complication, independently of infarct location (1-7).

How thrombolytic therapy affects the incidence and the prognosis of CAVB complicating AMI is not well established. On the one hand, some reports suggest that the

development of CAVB after thrombolysis might be caused by successful reperfusion (8). For example, in the TAMI (Thrombolysis and Angioplasty in Myocardial Infarction) study (9), CAVB was precipitated by an active component of reperfusion (thrombolysis, PTCA [percutaneous transluminal coronary angioplasty] or reocclusion) in 38% of patients. On the other hand, other studies hypothesized that the incidence and mortality from CAVB is expected to be reduced because thrombolytic therapy decreases infarct size (10,11). The incidence, in-hospital, and long-term effects of CAVB complicating AMI have not yet been reported from a large cohort of consecutive patients in the thrombolytic era.

Most of the studies, in both eras, compared AMI patients with and without CAVB. One study only (12) compared the outcome of patients with CAVB in the pre- and the

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Abbreviations and Acronyms

ACE	=	angiotensin-converting enzyme
AMI	=	acute myocardial infarction
CAVB	=	complete atrioventricular block
CCU	=	coronary care unit
CI	=	confidence interval
CK	=	creatinine kinase
HR	=	hazard ratio
OR	=	odds ratio
PTCA	=	percutaneous transluminal coronary angioplasty
RR	=	relative risk
SPRINT	=	Secondary Prevention Reinfarction Israeli Nifedipine Trial
TAMI	=	Thrombolysis and Angioplasty in Myocardial Infarction
TIMI	=	Thrombolysis in Myocardial Infarction trial

early thrombolytic (1986 to 1988) eras, reporting similar in-hospital survival associated with CAVB in both periods.

The aim of this study was to analyze the incidence, short-term and one-year prognosis of patients developing CAVB in the thrombolytic era and to compare it to data from the prethrombolytic era, in two large Israeli cohorts of consecutive patients with AMI.

METHODS

The first cohort of 3,300 unselected, consecutive patients with AMI was derived from two prospective national surveys, conducted during January to February 1992 and 1996 and May to July 1996 in all 25 coronary-care units operating in Israel (the thrombolytic era).

For comparison we used data from a second cohort of 5,788 consecutive patients with a confirmed AMI who were admitted to 13 coronary-care units operating between August 1981 and July 1983, in Israel. The patients were screened for inclusion in the Secondary Prevention Reinfarction Israeli Nifedipine Trial (SPRINT) (13) and included in the SPRINT Registry.

The diagnostic criteria for AMI (13), infarct size estimated by enzyme levels and infarct location were similar in both periods. Diagnosis of AMI was based on the presence of any two of the following criteria: typical chest pain lasting at least 30 min; new electrocardiographic (ECG) changes (Q/QS and/or ST-segment and T-wave changes), or rise of at least two of the three serum cardiac enzymes (creatinine kinase, aspartate aminotransferase, and lactate dehydrogenase) to more than 1.5 times the upper limit, or concomitant rise of creatine kinase (CK) and MB isoenzyme. The AMI location was determined by the Minnesota Code (14) as follows: anterior (V_1 through V_5), inferior (L_2 , L_3 or aVF), lateral (L_1 , aVL or V_6) and undetermined if complete left bundle branch block concealed the site of infarction.

The thrombolytic surveys and the SPRINT Registry files included demographic and medical data of patients from the

index hospitalization, medical history, in-hospital course and complications. Pharmacological therapy and interventional procedures performed during the index hospitalization were recorded for both periods. During the survey period in 1992 and 1996, we employed a conservative strategy of "watchful waiting" where coronary angiography followed by mechanical reperfusion was used only for patients with spontaneous or provocative ischemia (15).

Complete heart block was defined as a third-degree atrioventricular block when no atrial activity was conducted to the ventricles (16) and a ventricular rate of ≤ 50 beats/min.

All medical summary reports of the patients with the diagnosis of CAVB were reviewed. Fifteen reports were not available for review (12/356; 3.4%) from the prethrombolytic era and 3/140 reports (2.1%) from the 1990s, and these patients were excluded.

To avoid inclusion of patients with CAVB in whom such an event was not part of the "natural history" of AMI complicated by CAVB, patients in whom CAVB occurred as a terminal event were also excluded. Therefore, exclusion criteria were 1) CAVB developing as a terminal rhythm in the setting of cardiogenic shock or pump failure developing during hospitalization in the coronary care unit (CCU). Patients who presented with CAVB and cardiogenic shock on admission were included; and 2) transient CAVB occurring during mechanical revascularization. Fifty-four such patients (39/356 [10.9%] in the 1980s and 15/140 [10.7%] in the 1990s) were therefore excluded.

Mortality rates at seven days, 30 days and one year in both study periods were assessed from medical charts and by matching the identification numbers of the patients with the Israeli National Population Register.

Statistical analysis. Statistical analysis was performed using SAS software. The chi-square test and *t* test were used to determine the significance of the differences between proportions and means, respectively. Results of continuous variables are reported as mean \pm SD. A *p* value ≤ 0.05 was considered statistically significant.

To compare mortality in the two periods (1992 to 1996 vs. 1981 to 1983), the adjusted OR with 95% CI for seven- and 30-day mortality was calculated using the LOGISTIC procedure (17). Multivariate analysis of one-year and 30-day to one-year mortalities was performed using the Cox proportional hazard model (PHREG procedure) (18). Adjustment was made for age, gender, history of diabetes mellitus, hypertension, angina, previous AMI, congestive heart failure on admission, and anterior infarct location. A similar method of analysis was used to compare mortality in patients with and without CAVB in 1992 and 1996. Logistic regression analysis was used to determine significant predictors of CAVB in the thrombolytic era.

Survival curves were constructed using the Kaplan-Meier method. The significance of the differences between the survival curves was assessed by the log-rank test (SAS LIFETEST Procedure).

Table 1. Parameters Predicting Development of CAVB in the Thrombolytic Era

Premature	OR of CAVB	95% CI
Age (10 yr increment)	1.16	0.98-1.38
Female gender	1.72	1.14-2.59
Anterior infarction	0.11	0.06-0.20
Killip class ≥ 2	2.88	1.88-4.38
Thrombolytic therapy	2.06	1.38-3.08
Past angina	0.61	0.40-0.91

Stepwise from age, gender, anterior AMI, previous AMI, Killip ≥ 2 , thrombolytic therapy, diabetes mellitus, hypertension, past angina.

CAVB = complete atrioventricular block; CI = confidence intervals; OR = odds ratio.

RESULTS

Patients in the thrombolytic era: CAVB versus non-CAVB. The frequency of CAVB in the thrombolytic era was 3.7% (122/3,300). Patients who developed CAVB in 1992 and 1996 were older (66 ± 12 vs. 63 ± 13 years, $p = 0.005$), included more women (40% vs. 25%, $p < 0.001$) and, as expected, had a lower incidence of anterior infarct location (11% vs. 45%, $p < 0.0001$) and non-Q-wave AMI (10% vs. 23%, $p < 0.002$) than did those without CAVB.

A multivariate analysis performed to identify independent risk factors for the occurrence of CAVB revealed that female gender, treatment with thrombolysis and a Killip class ≥ 2 at presentation were associated with an increased risk for the development of CAVB (Table 1). Anterior location of infarct and a history of angina pectoris were inversely associated with CAVB.

In the thrombolytic era, seven-day (21% vs. 6%), 30-day (29% vs. 10%), and one-year cumulative crude mortality rates (35% vs. 15%) ($p < 0.00001$ for each) were higher among patients who experienced CAVB than in those who did not (Fig. 1). These differences in prognosis persisted also after multivariate adjustment (Table 2); 7-day OR =

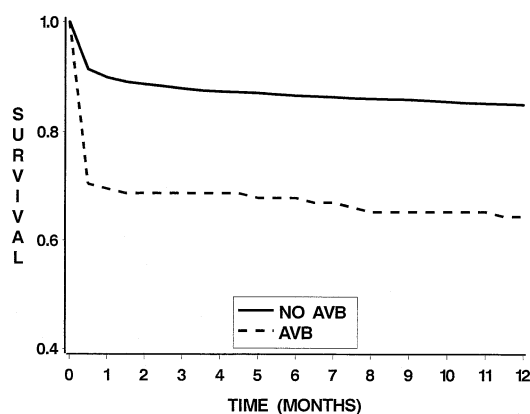


Figure 1. One-year survival curves for patients with and without CAVB complicating AMI in the thrombolytic surveys (p by the log-rank test = 0.0001).

Table 2. Odds Ratios and Hazard Ratios of Mortality of Patients With CAVB Complicating AMI

OR*/HR† (95% CI)	1992-1996 vs. 1981-1983	1992-1996 CAVB vs. non-CAVB
7-day	0.82 (0.46-1.43)	4.05 (2.34-6.82)
30-day	0.78 (0.45-1.33)	3.98 (2.44-6.43)
30-day to 1-year	0.49 (0.18-1.31)	0.93 (0.41-2.14)
1-year cumulative	0.79 (0.54-1.56)	2.36 (1.68-3.30)

*Odds ratio for 7- and 30-day or †hazard ratio for 30-day to one-year and one-year cumulative mortality. Adjusted for age, gender, diabetes mellitus, hypertension, angina, previous infarction, heart failure on admission and anterior infarct location.

4.05 (95% CI 2.34 to 6.82), 30-day OR = 3.98 (95% CI 2.44 to 6.43) and one-year HR = 2.36 (95% CI 1.68 to 3.30). Therefore, CAVB emerged as an independent predictor of mortality in the thrombolytic period.

One-year mortality among 30-day survivors was similar in those with and without CAVB (HR = 0.93, 95% CI 0.41 to 2.14).

Patients with CAVB in the thrombolytic era: Thrombolysis versus non-thrombolysis. During the 1992 and 1996 surveys, 1,567/3,300 patients (47.5%) were treated with thrombolytic agents (Table 3). A trend toward a higher crude incidence of CAVB was observed in patients treated (68/1,567, 4.3%) versus not treated with thrombolysis (54/1,733, 3.1%; $p = 0.07$). To exclude selection bias of patients with CAVB to either therapy, the causes for excluding patients from thrombolysis were analyzed. Late arrival (suggesting underestimation of the incidence of early occurrence of transient CAVB) and previous treatment with beta-blockers (thus increasing the likelihood for developing CAVB) (12), which might be possible causes for excluding patients from thrombolysis, were equally distributed in those who developed CAVB and those who did not. Other reasons for ineligibility for thrombolysis (i.e., bleeding tendency, neoplastic disease, a recent cerebrovascular event) were similarly distributed between the two groups as well.

Patients who were treated with thrombolytic therapy and developed CAVB were younger (62 vs. 71 years, $p = 0.0001$) and had a lower incidence of a previous myocardial infarction, cerebrovascular accident and diabetes mellitus and a higher incidence of smoking history than did counterparts who did not undergo thrombolysis, reflecting the differences in baseline characteristics between all patients being treated with thrombolysis and those not being treated.

A multivariate analysis performed to identify independent risk factors for the occurrence of CAVB revealed that thrombolytic therapy was independently associated with a twofold increased risk for the development of CAVB (Table 1).

Thirty-day mortality among inferior AMI patients with CAVB was significantly higher than in counterparts without CAVB, irrespective of whether they received (CAVB vs. no-CAVB: 22% vs. 5%, respectively, $p < 0.001$) or did not

Table 3. Baseline Characteristics of Patients With CAVB Complicating AMI

%	SPRINT 1981–1983 (n = 5,788)		Thrombolytic Surveys 1992 and 1996 (n = 3,330)			
	All CAVB (n = 305)	p Value*	All CAVB (n = 122)	Thrombolysis (n = 68)	p Value†	No Thrombolysis (n = 54)
CAVB	5.3	0.0007	3.7	4.3	0.07 (NS)	3.1
Age (yrs) (mean ± SD)	66 ± 10	NS	66 ± 12	62 ± 12	0.0001	71 ± 12
Male	68	NS	60	65	NS	55
<i>History</i>						
MI	24	NS	20	10	0.004	32
Angina pectoris	49	0.001	31	34	NS	28
Hypertension	47	NS	41	37	NS	46
Diabetes mellitus	19	0.04	28	21	0.04	37
Cerebrovascular accident	5	NS	4	0	0.01	9
Current smokers	28	NS	34	47	0.001	19
<i>Infarct location</i>		0.002			NS	
Anterior	27		11	12		11
Inferior/Posterior	73		88	88		88
Lateral	0		1	0		1
Undetermined	0		0.4	0		0
Non-Q-wave MI	4	0.02	10	7	NS	16

*Comparing patients with CAVB in 1981–83 and 1992 and 1996 cohort. †Comparing thrombolized and nonthrombolized patients with CAVB in 1992–1996.
CAVB = complete atrioventricular block; MI = myocardial infarction; NS = nonsignificant.

receive thrombolytic therapy (CAVB vs. no-CAVB: 35% vs. 8%, respectively, $p < 0.001$). Similar findings were observed in patients with anterior AMI who were *not* treated with thrombolysis: mortality among patients with CAVB was significantly higher than in counterparts without CAVB: 67% vs. 16%, respectively, $p = 0.007$. Thirty-day mortality among anterior AMI patients who were treated with thrombolysis did not differ between patients with CAVB and counterparts without CAVB (14% vs. 9%, respectively, $p = \text{NS}$). However, the number of patients with CAVB complicating anterior AMI was small—13 patients—so conclusions from such numbers should be interpreted with caution.

Seven-day crude mortality among patients with CAVB who were treated by thrombolytic therapy in 1992 and 1996 was similar to that of counterparts not treated with thrombolysis, whereas late mortality (30 day and one year) was

lower among thrombolysis-treated patients. (Table 4 and Fig. 2). However, after adjustment, no difference in mortality was observed between the two groups of patients with CAVB: the 7- and 30-day OR were 0.82 (95% CI 0.30 to 2.26) and 0.49 (95% CI 0.20 to 1.17), respectively, and the one-year HR was 0.87 (95% CI 0.41 to 1.84).

Patients with CAVB: 1992 and 1996 versus 1981–1983.

The frequency of CAVB complicating AMI decreased significantly in the thrombolytic era as compared with a decade earlier (3.7% vs. 5.3%, $p = 0.0007$) (Table 3). Baseline characteristics of patients with CAVB were similar in both periods, except for a higher incidence of a history of angina and a lower incidence of diabetes mellitus among patients included in the 1981 to 1983 cohort (Table 3). The incidence of CAVB was even higher when comparing the prethrombolytic cohort with the group of patients in the

Table 4. Crude Mortality Rates of Patients With CAVB Complicating AMI

	SPRINT 1981–1983 (n = 5,788)		Thrombolytic Surveys 1992 and 1996 (n = 3,300)			
	All CAVB (n = 305)	p Value*	All CAVB (n = 122)	Thrombolysis (n = 68)	p Value†	No Thrombolysis (n = 54)
<i>Mortality (%)</i>						
7 day	31	0.04	21	16	NS	28
30 day	42	0.01	29	21	0.02	40
30 day to 1 year	14	NS	7	6	NS	10
1-year cumulative	50	0.005	35	26	0.01	48

*By univariate analysis between the 1981–1983 and 1992 and 1996 cohorts. †By univariate analysis between thrombolized and nonthrombolized patients.

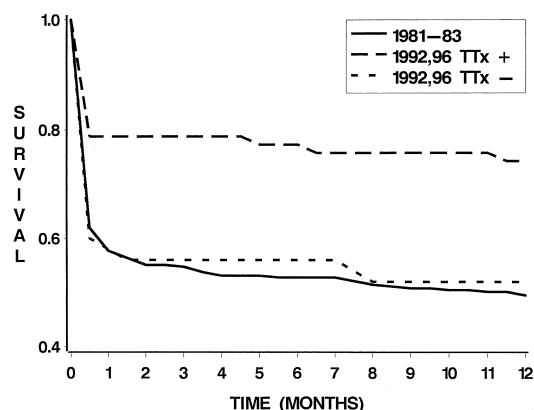


Figure 2. One-year survival curves for patients with CAVB complicating AMI in the prethrombolytic and thrombolytic eras. **TTx (+)** = thrombolytic therapy; **TTx (-)** = no thrombolytic therapy. When comparing the thrombolysed groups in 1992 and 1996 with the 1981 to 1983 cohort, $p = 0.006$.

1990s not treated with thrombolysis (5.3% vs. 3.1%, $p = 0.0001$), despite worse clinical predictors of mortality in the latter group.

Infarct location among patients who developed CAVB differed between the two cohorts; anterior location was less prevalent and inferior/posterior location was more prevalent in the thrombolytic era as compared with the prethrombolytic era ($p = 0.002$) (Table 3).

In addition, no significant reduction in one-year mortality was observed in the thrombolytic era among patients with both anterior (54% vs. 75%, $p = \text{NS}$) and inferior (32% vs. 41%, $p = \text{NS}$) sites of infarction, compared with the prethrombolytic era.

Timing of the occurrence of CAVB was evaluated. This complication was observed on hospital admission in 55% and in 73% of patients with CAVB (for whom timing was reported), in the 1980s and 1990s, respectively. Within the first 48 h, an additional 34% and 21% of patients in the prethrombolytic and the thrombolytic eras, respectively, developed CAVB. Thus, 87% and 94% of CAVBs occurred early in the course of AMI.

Seven-, 30-day and one-year crude mortality rates in patients with CAVB were relatively high but lower in the thrombolytic era in comparison to the prethrombolytic era (21%, 29% and 35% vs. 31%, 42% and 50%, respectively) (Table 4 and Fig. 2). However, following multivariate analysis, the adjusted risk of mortality was similar to that in the previous decade (Table 2); 7-day OR = 0.82 (95% CI 0.46 to 1.43); 30-day OR = 0.78 (95% CI 0.45 to 1.33) and one-year HR = 0.79 (95% CI 0.54 to 1.56).

Moreover, a significant reduction in 30-day mortality was observed among men with CAVB in the thrombolytic era in comparison to the prethrombolytic era (24% vs. 37%, $p < 0.04$). No significant reduction in mortality was observed in patients with first or recurrent AMI, anterior or inferior sites of infarction in both periods.

The one-year outcome of 30-day survivors with CAVB during hospitalization in 1992 and 1996, was not significantly different from that of patients in 1981 to 1983: 7% versus 14% ($p = \text{NS}$), HR = 0.49, 95% CI 0.18 to 1.31).

DISCUSSION

To our knowledge, this is the first population-based, multicenter, nationwide study of CAVB complicating AMI in the thrombolytic era.

The present study shows a significant decrease in the occurrence of CAVB after AMI in the thrombolytic (3.7%) as compared to the prethrombolytic era (5.3%). Despite this improvement, adjusted mortality among patients with CAVB has not changed within the last decade. Mortality of patients with CAVB remained higher than that of patients without CAVB. Similar to observations from the prethrombolytic era, the occurrence of CAVB during AMI in the 1990s had no impact on one-year mortality among 30-day survivors.

Incidence. The incidence of CAVB in the thrombolytic era was reduced compared to the prethrombolytic era in accordance with most (3,5,19,20) but not all (12) previous studies. Interestingly, the incidence of CAVB among our patients with inferior myocardial infarction (6.0%) is lower than that in the TAMI (Thrombolysis and Angioplasty in Myocardial Infarction) (13%) and TIMI (Thrombolysis in Myocardial Infarction) (12%) studies (9,21). This could be explained by the differences between the populations: our study is a nationwide study that includes the entire AMI population, whereas the TIMI and TAMI studies include a highly selected population referred to tertiary hospitals for thrombolysis. In addition, all patients in the TIMI and TAMI trials received thrombolysis; in our study, only 47% of patients were treated with lytic therapy.

The observed reduced incidence of CAVB among patients with both anterior and inferior infarction in 1992 and 1996 versus the previous decade may be related to enhanced coronary perfusion and myocardial revascularization in the thrombolytic era, thus limiting infarct size and preserving ventricular function (10,11). The reduced incidence of CAVB, also observed among nonthrombolysed patients, probably reflects an overall change in management of all AMI patients treated today, as has recently been shown, both in Europe (22) and North America (23,24).

Studies from the prethrombolytic era report the presence of CAVB in up to 55% of patients developing this complication, upon hospital admission and in up to 75% within 24 h (2,3,19,20), similar to the TIMI (21) and TAMI (9) trials in which CAVB was with this event present in 53% and 54% of cases with this event, respectively, upon admission and in 96% within 72 h. These figures are in accordance with the data reported in our study: 87% of cases in the prethrombolytic and 94% in the thrombolytic eras, respectively, of all CAVB cases appear within 48 h after admission.

Pathogenesis. The CAVB complicating anterior AMI is usually within the HIS-Purkinje system and is related to interruption of septal perfusion accompanied by extensive myocardial damage and significant left ventricular dysfunction (3,4). In inferior AMI, CAVB usually involves the supra-Hissian atrioventricular junction (25) due to hypoperfusion of the atrioventricular nodal artery.

The reperfusion of the infarct-related artery should conceivably reduce the incidence of CAVB in both anterior and inferior infarctions. Surprisingly, we found that the incidence of CAVB was not reduced by thrombolytic therapy and the additional procedures aimed at infarct and ischemic area reduction. Furthermore, thrombolytic therapy was found to be an independent risk factor for developing CAVB. The reason for this interesting finding is unknown. It might be due to association—patients with higher ST-segment elevation are more likely to have a larger AMI, therefore more likely to receive thrombolysis. Such patients might be more prone to develop CAVB. One possible mechanism may be vagally mediated CAVB, which has been suggested as a sign of successful reperfusion (8). This results from restoration of flow that facilitates leukocyte migration to the infarcted area, stimulating vagal innervation within the infarcted myocardium (26). Additionally, CAVB may occur as a consequence of reocclusion following successful reperfusion (occurring in 14% of CAVB patients in the TAMI trial) (9). Finally, reperfusion injury may further hamper the conduction system; reperfusion was a precipitating event in 10% of CAVB patients in the TAMI trial (9). In the latter study, CAVB was associated with “an active component of reperfusion” in almost 40% of patients.

In our study, a history of angina pectoris was negatively associated with the development of CAVB, in accordance with a recent study suggesting that absence of preinfarction angina predicted the development of CAVB (27). Preinfarction angina is accompanied by a smaller infarct size, preservation of left ventricular function (preconditioning) and a higher and more rapid patency of the infarct-related artery (28,29), therefore reducing the occurrence of periatrioventricular nodal ischemia.

Mortality. In the present study, patients who developed CAVB after AMI showed no significant reduction of the adjusted short-term and one-year mortality, compared to the prethrombolytic era.

Patients with CAVB during AMI have an increased in-hospital mortality and fare worse than do counterparts with no CAVB (3,4,6). Their prognosis is particularly poor during hospitalization (2,7,9,20), in both the prethrombolytic (7,30) and the thrombolytic eras (9,31). Similar observations were made in the present study; the seven- and 30-day mortality was about four times higher and the one-year mortality was twice as high among patients with CAVB than among counterparts who did not develop this complication.

Previous studies have shown that the immediate poorer

prognosis of AMI patients with CAVB results from larger infarctions (32) with a correlation between infarct size and the occurrence of CAVB in both anterior and inferior infarctions (33). Apparently, CAVB is not responsible for the increased mortality (20), but rather is a marker for an increased infarct size. In addition, CAVB was independently associated with increased in-hospital mortality, both in the previous decade (7) and in the 1990s (9). Our previous study (7) reported a mortality rate four times higher among anterior AMI patients with CAVB than among counterparts without CAVB. We (20) and others (2,5,9) also reported a higher mortality among inferior AMI patients with CAVB than among those with no CAVB (RR = 2.0).

The frequent resolution of CAVB in AMI survivors is related to spontaneous (34) or medical (35) reperfusion, resulting in disappearance of atrioventricular nodal ischemia. Mortality among 30-day survivors in the 1990s was not affected by the occurrence of CAVB as we (7,20) and others (12,36,37) previously reported.

Mortality among inferior AMI patients with CAVB was similar among those treated and not treated with thrombolysis, despite better baseline characteristics in the former. This could be attributed to the deleterious effect of the occurrence of CAVB on prognosis, which counterbalances the theoretical benefit that could have been achieved with thrombolysis. The presence of CAVB could possibly reduce the benefit of thrombolytic therapy because of the associated bradycardia and hypotension. This hypothesis is supported by a higher incidence of TIMI-0 flow in the atrioventricular-block group (31).

Survival in nonthrombolized patients with CAVB in 1992 to 1996 was not better than in 1981 to 1983. This may reflect the effects of two opposing factors: nonthrombolized patients with CAVB today have worse baseline characteristics in comparison to CAVB patients a decade ago (Table 3); however, improvement in drug therapy (i.e., increased use of aspirin, beta-blockers and ACE [angiotensin-converting enzyme] inhibitors) contributes to reduced current mortality (22–24).

Study limitations. The SPRINT Registry and the 1992 to 1996 cohort were derived from prospective nationwide surveys, which were not designed to address patients with CAVB specifically. Thus, no data on duration of CAVB or its relation to initiation of thrombolytic therapy were available.

The use of historical controls is a limitation because of the changes in therapy that occurred during the last decade, such as ACE inhibitors, aspirin, beta-blockers, and mechanical revascularization. However, although such interventions were expected to reduce mortality in the 1990s, this has not changed significantly between the two periods.

Conclusions. The incidence of CAVB in the general AMI population declined in the thrombolytic era compared to the previous decade. However, CAVB is still accompanied by a

poor early prognosis, whereas the long-term outcome of hospital survivors is similar to patients who did not experience CAVB during hospitalization. A more aggressive therapeutic approach aimed to reduce early mortality seems warranted in these patients.

APPENDIX 1

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APPENDIX 2

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